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AUDITORY EVENT–RELATED DYNAMICS OF THE EEG

SPECTRUM AND EFFECTS OF EXPOSURE TO TONES

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Auditory event-related dynamics of the EEG spectrum and effects of exposure to tones *

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Summary   A new measure of event-related brain dynamics, the event-related spectral perturbation (ERSP), is introduced to study event-related dynamics of the EEG spectrum induced by, but not phase-locked to, the onset of the auditory stimuli. The ERSP reveals aspects of event-related brain dynamics not contained in the ERP average of the same response epochs. Twenty-eight subjects participated in daily auditory evoked response experiments during a 4 day study of the effects of 24 h free-field exposure to intermittent trains of 89 dB low frequency tones. During evoked response testing, the same tones were presented through headphones in random order at 5 sec intervals. No significant changes in behavioral thresholds occurred during or after free-field exposure. ERSPs induced by target pips presented in some inter-tone intervals were larger than, but shared common features with, ERSPs induced by the tones, most prominently a ridge of augmented EEG amplitude from 11 to 18 Hz, peaking 1–1.5 s e after stimulus onset. Following 3–11 h of free-field exposure, this feature was significantly smaller in tone-induced ERSPs; target-induced ERSPs were not similarly affected. These results, therefore, document systematic effects of exposure to intermittent tones on EEG brain dynamics even in the absence of changes in auditory thresholds.

Key words: EEG spectrum; auditory evoked response; Temporary threshold shift

Limitations of the event-related potential (ERP)

Most studies of brain electrical activity recorded from the scalp are concerned either with analysis of differences in the mean spontaneous EEG frequency spectrum under various conditions, or with measures on averages of precisely time-locked brain responses evoked by discrete experimental stimuli or events. The low amplitude of most evoked response components recorded from the scalp makes study of their parameters in single trials difficult or impossible. By averaging response epochs time-locked to a number of similar events, rhythmic or quasi-rhythmic brain activity not exactly synchronized in both time and phase to the experimental events is averaged out through phase cancellation, and the coherent time- and phase-locked activity that remains is called an event-related potential (ERP). Since the development of computer averag-

ing technology in the 1960s, the average evoked response method has become predominant for human experiments.

However, the electrophysiological response of the brain to auditory events is not fully captured by the ERP. This would only be the case if (1) evoked responses were stable across trials, (2) were completely independent of the ongoing EEG, and (3) the ongoing EEGs were not itself perturbed by experimental events. Several reports have shown that ERPs are neither stable nor wholly independent of the EEG (e.g., Başar 1980; Jansen and Brandt 1991) and, therefore, the response of the brain to auditory or other experimental events is not fully captured in the ERP.

Efforts to observe, quantify, and best characterize phase in coherent event-related brain dynamics is an emerging research topic (Achimowicz 1992; Pfurtscheller 1992; Van Dijk et al. 1992). Violations of independence of the EEG and ERP can be modeled either as effects of experimental events on the EEG and/or as non-stationarity in the ERP itself. That is, the difference between any single response epoch and the average ERP may be modeled either as being a perturbed continuation of pre-baseline EEG activity, as variance in the evoked response, or as some combination of these.

The event-related spectral perturbation (ERSP)

In modeling event-related reactivity in the ongoing EEG, amplitude and phase effects may be considered separately or in combination. A well-known example of an event-related EEG amplitude change is the reduction in occipital alpha band EEG amplitude that normally occurs when the eyes are opened. The time course of event-related attenuations in alpha band EEG was first quantified by Pfurtscheller (1977), using a term, event-related desynchronization (ERD), derived from the concept that cortical arousal involves desynchronization of relatively slow, spatiotemporally coherent cortical rhythms, and their replacement with faster, more spatially differentiated activity. ERD analysis was first used to quantify the degree to which the amplitude of prominent ongoing ("synchronized") alpha band EEG was depressed (or "desynchronized") following arousing events in an auditory contingent negative variation (CNV) paradigm, the time course and scalp topography of this depression depending on the stimuli and subject task and conditions (Pfurtscheller and Klimesch 1991). Recently, Pfurtscheller (1992) has proposed the term event-related synchronization for time-locked amplitude enhancement, and Van Dijk et al. (1992) have analyzed background reactivity in various pre-defined frequency bands.

The measure introduced here, the event-related spectral perturbation (ERSP), can be viewed as a generalization of the ERD. The ERSP measures average dynamic changes in amplitude of the broad-band EEG frequency spectrum as a function of time relative to an experimental event. That is, the ERSP measures the average time course of relative changes in the spontaneous EEG amplitude spectrum induced by a set of similar experimental events. These spectral changes typically involve more than one frequency or frequency band, so full-spectrum ERSP analysis yields more information on brain dynamics than the narrow-band ERD.

To compute an ERSP, baseline spectra are calculated from the EEG immediately preceding each event. The epoch is divided into brief, overlapping data windows, and a moving average of the amplitude spectra of these is created. Each of these spectral transforms of individual response epochs are then normalized by dividing by their respective mean baseline spectra. Normalized response transforms for many trials are then averaged to produce an average ERSP, plotted below as relative spectral log amplitude on a time-by-frequency plane.

The current study

Prolonged exposure to intense sound can produce temporary or permanent hearing loss and other physiological effects (Clark 1991; Jansen 1991), as well as emotional stress and cognitive perf ⋯. ⋅e decre-

ments (Dejoy 1984; Jones and Broadbent 1991). Pathological effects of noise are often assumed to begin with temporary exposure-related increases in thresholds, termed temporary threshold shifts (TTS) (Melnick 1991). These are accompanied by changes in auditory nerve activity and in auditory brain-stem responses (ABRs) recorded from the scalp (Attias and Pratt 1985). However, the effects of sound exposure on later evoked response features and on the EEG spectrum do not seem to have received much attention. This paper explores the effects of sound exposure on patterns of auditory event-related EEG spectral reactivity. It reports results of an experiment conducted during a larger study of the effects of exposure to active sonar signals (Coyne 1992) investigating the effects on EEG spectral reactivity of 24 h of intermittent free-field exposure to low frequency tones delivered at a level and duty cycle insufficient to induce permanent or temporary threshold shifts in any subject. Results demonstrate that characteristic event-related perturbations of EEG spectral amplitudes (ERSPs) are induced by both tones and tone pips. Prolonged exposure to tones are found to produce changes in this auditory spectral reactivity, even in the absence of behioral threshold shifts.

Methods

Subjects

Four groups of 10–12 normal hearing young adult volunteers (ages 19–34 years) were housed for 4 days in an enclosed laboratory setting. On each day, a fixed schedule of experiments was conducted, consisting of repeated audiograms, computerized cognitive tests, and physiological measures including the da.iy evoked response experiment reported here. Data from the 28 subjects who completed all portions of the experiment were used in the final analysis.

Exposure

Beginning at 8 a.m. of the third day, and continuing for 24 h until 8 a.m. of the fourth day, trains of 6 consecutive 1 sec pure tones were delivered in the free-field of the subjects' living quarters via multiple loudspeakers with an inter-train interval of 24 sec. Tone frequencies were 720, 800, 880, 960, 1040 and 1120 Hz. Each tone had a linear 25 msec onset and offset. During exposure 6 different trains consisting of these 6 frequencies in different orders were presented continuously, at 30 sec intervals, in cyclic order. Intensity of free-field stimulation was 89 dB (A-weighting). Free-field sound intensities were made uniform by careful balancing of multiple loudspeakers (Krebs et al. 1992). Subject dosages were verified by audio dosimeters worn by some of the subjects. Free-field exposure

occurred only on day 3; on days 1, 2 and 4, no free-field stimulation was given.

Testing procedure

Each of the 4 days of the session, subjects participated in a 15 min experiment in which the same 1 sec tones delivered in the free-field were delivered to the subject binaurally via headphones at 89 dB. Subjects were tested one at a time, in the same order each day, the experiments lasting from about 11 a.m. to 6 p.m. Each day at the time of testing, subjects would first be fitted with EEG electrodes. Then they would leave their free-field exposure living quarters and walk 50 feet to the evoked response testing chamber. Testing would begin within a few minutes after arrival.

Test stimuli

During the experiment, 3 concurrent streams of auditory stimuli were delivered to subjects: the 1 sec tones; occasional brief, high-frequency tone pips which served as target stimuli; and a continuous steady-state stimulus train at 40 dB normal hearing level (nHL).

These are illustrated in Fig. 1. The tones were identical to the tones used in free-field exposure. However, because of technical limitations, instead of being delivered in trains of 6 tones in succession as in the free-field stimulation, during evoked response testing the same 1 sec tones were presented individually, in pseudo-random order, with a stimulus onset asynchrony (SOA) of 5 sec. This resulted in the same mean presentation rate (12 tones/min) or duty cycle (20%) as in the free-field stimulation.

The target pips had a duration of 25 msec and a frequency of 3000 Hz. They were delivered in pseudo-random order and intervals in one-sixth of the inter-tone intervals at 95 dB (A-weighting), yielding a mean rate of 2 targets/min. Subjects were instructed to keep their eyes closed and to press a response button each time they heard a target pip. The steady-state stimulus train consisted of 980 Hz tone pips (5 msec cosine squared envelope) at 40 dB nHL with a repetition rate of one-eighth the EEG sampling rate. Its purpose was to drive a steady-state response (SSR) at the 39 Hz stimulus rate (Galambos et al. 1981), to test whether changes during exposure could be detected in the SSR.
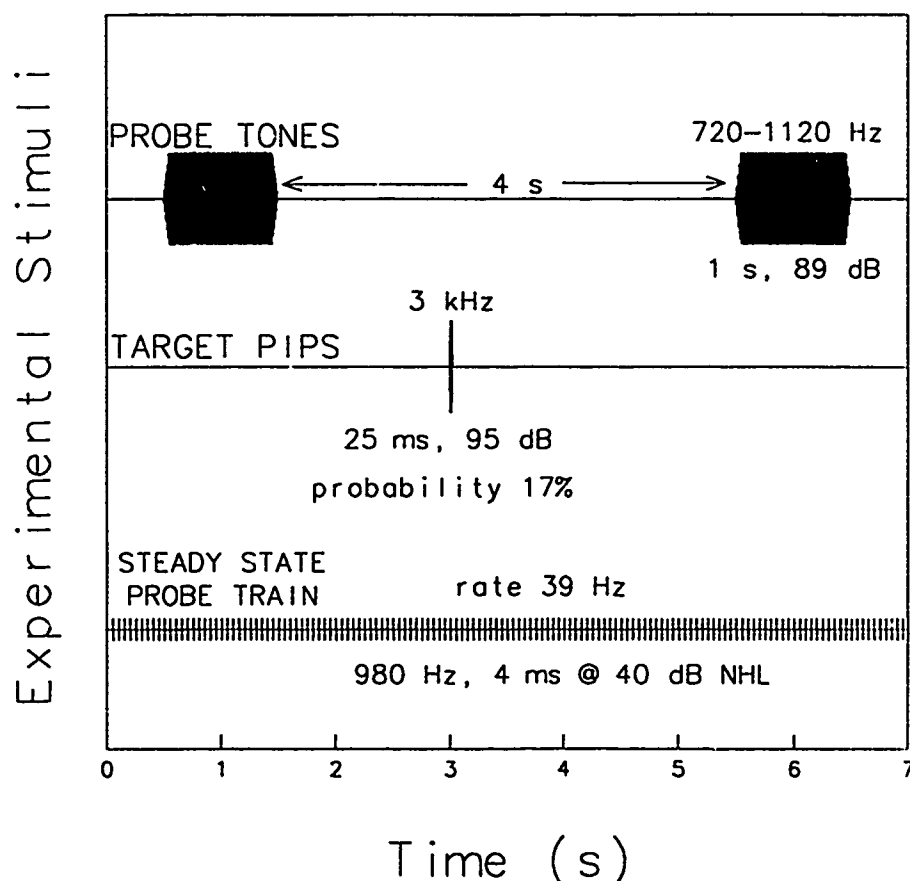

Fig. 1. Schematic view of the 3 streams of test stimuli used in the experiment. The top trace shows 1 sec tones which are presented at regular 5 sec intervals. Tones of 6 frequencies are delivered in pseudo-random order. In one-sixth (17%) of the trials, a brief, high-frequency tone pip (middle trace) is presented at a random point during the inter-tone interval. Subjects press a response button whenever the target pip is presented. In the background (bottom trace), a continuous 39 Hz steady-state response probe train is presented throughout the experiment.
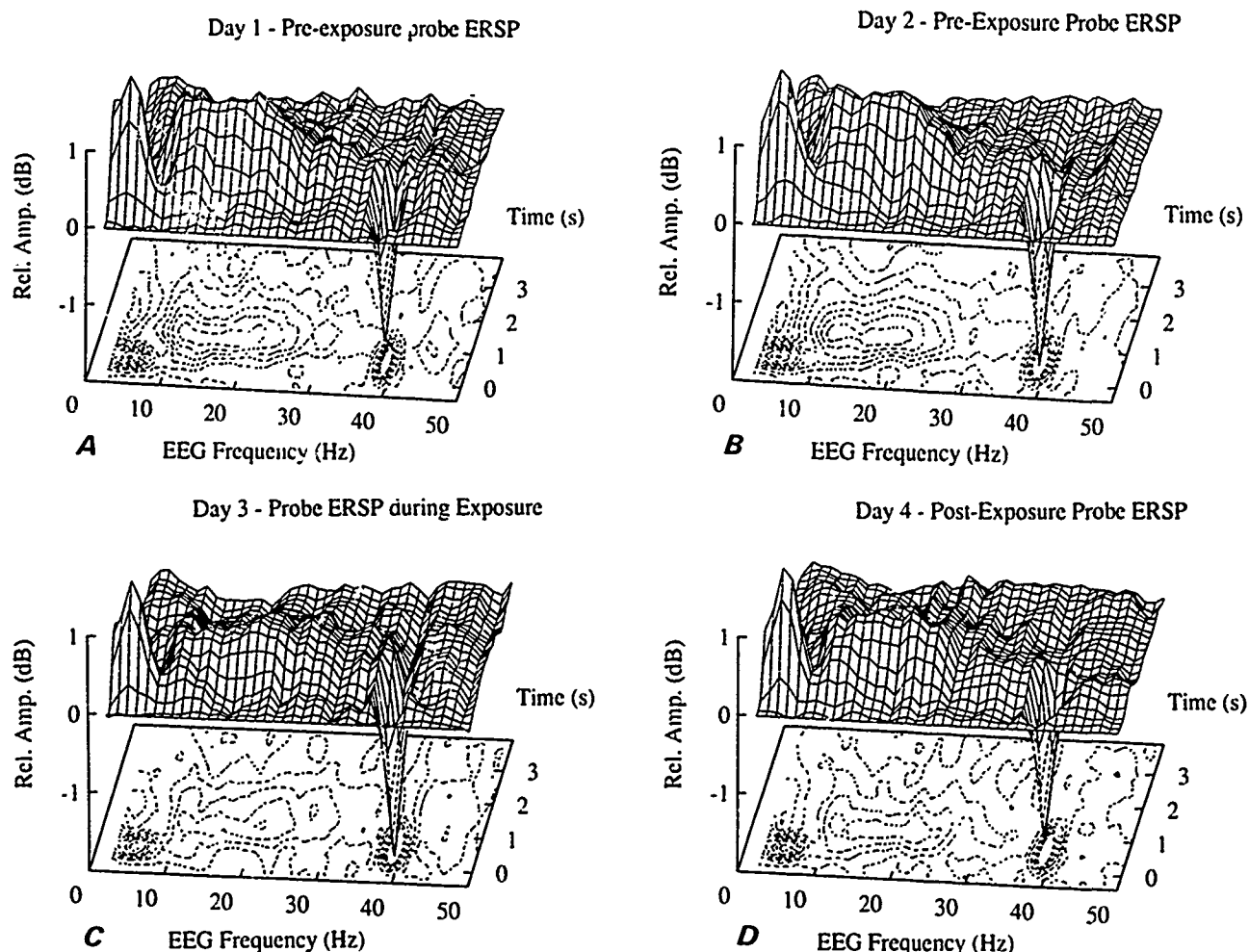
Day 1 - Pre-exposure probe ERSP

Day 2 - Pre-Exposure Probe ERSP

Day 3 - Probe ERSP during Exposure

Day 4 - Post-Exposure Probe ERSP

Fig. 2. ERSPs induced by 1 sec task-irrelevant tones on days 1–4; grand means of responses from 28 subjects for all 6 tone frequencies (720–1120 Hz). In each plot, the left-right axis is EEG frequency; the front-back axis is time relative to tone onset (at time 0); the vertical axis is EEG amplitude ratio plotted in (20 $\log_{10}$) dB. A, B: the mean ERSP for day 1 is closely replicated on day 2. C: note the smaller 11–18 Hz enhancement during free-field exposure on day 3, and its partial recovery in D after exposure on day 4.

Recording

EEG was collected from a scalp electrode located midway between Cz and Fz of the international 10–20 system, referenced to the left earlobe. Horizontal and lateral eye movements were also recorded. EEG recording was continuous, with a sampling rate of 312.5 Hz/channel using a 12-bit analog-to-digital converter and an analog passband of 0.1–100 Hz [1].

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[1] Subsequent to data collection, it was found that because of a minor computer malfunction, stimuli and EEG recording had not been precisely synchronized. Using steady-state response (SSR) phase results, it was determined that stimulus presentation lagged behind EEG data collection at a rate of one frame in 7728. Using this information, moments of stimulus onset were corrected post hoc. Because ERSP analysis trades time resolution for frequency resolution, remaining uncertainty in stimulus timing (estimated from control runs as less than 25 msec (root mean squared)) could affect the ERSP results very little. However, ERP measures depend on more precise response phase synchronization and, therefore, are not reported here.

Analysis

For each run, EEG epochs of 5.94 sec beginning 1.23 sec before the onset of those tones not followed by a target pip were selected from the continuous EEG records. After responses containing significant eye movement or muscle artifacts were rejected for averaging, ERSPs to each of the 6 tone frequencies were computed. To compute the ERSPs, each response epoch was submitted to short-time Fourier analysis using Fast Fourier Transforms (FFTs). Each epoch of 1856 points comprised 26 Hanning-windowed, 256-point data windows with 75% overlap. Output consisted of 129 amplitude estimates with a frequency bin width of 1.22 Hz.

To reduce random error, the 26 spectral estimates for each epoch were smoothed using a 3-window moving average, resulting in 24 smoothed estimates with a total spectral window length of 1.23 sec. Each moving-average spectral estimate was then normalized by dividing by the first smoothed spectral estimate, repre-
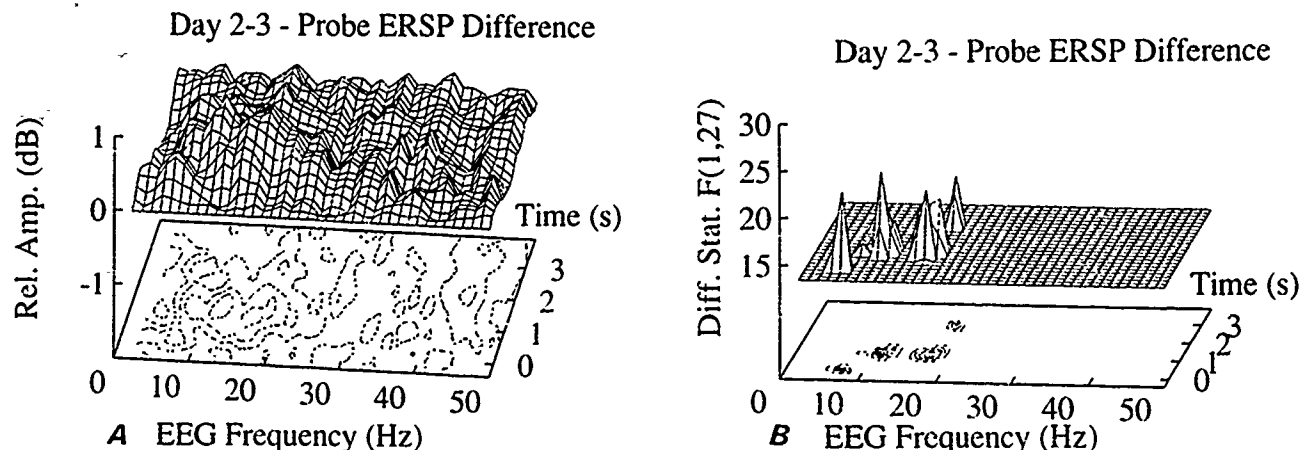
Fig. 3. A. difference between the tone-induced ERSPs for day 2 (prior to exposure) and day 3 (during exposure). Note the relative suppression of enhancement near 11 Hz, and the more sustained suppression of enhancement near 18 Hz on day 3. B. plot of F statistics, showing points of significant difference between tone-induced ERSPs for days 2 and 3, computed by ANOVA from responses of 28 subjects for each point in the time/frequency ERSP plane. Baseline: $F (1, 27) = 13.5$, $P = 0.001$.

senting the 1.23 sec pre-stimulus baseline period. Finally, a mean subject ERSP was computed for each run by averaging dB ($20 \log_{10}$) transforms of the individual epoch ERSPs for the 6 tones. The smoothing, normalization, and log averaging produced ERSP estimates of mean dB increase or decrease in spectral EEG amplitude time-locked to the stimulus [2].

Similar ERSP transforms of target stimulus-locked response epochs were also computed and averaged. To avoid overlap with ERSP activity to succeeding tones, only those targets presented at least 2.8 sec before the beginning of the next tone were used in the analysis. Target response epochs began 1.23 sec before and ended 2.8 sec after target onset. Targets not followed by a button press and target epochs containing gross muscle artifacts were rejected for averaging.

Results

Behavioral thresholds

Results of audiometric testing are more fully reported elsewhere (Krebs et al. 1992). No significant (≥ 5 dB) changes in threshold were found during or after exposure for any subject at any frequency tested. Nor was there any consistent change in thresholds with

increasing time of exposure, relative to pre-exposure measures. Thus the intensity and duty cycle of the sound the subjects were exposed to were not sufficient to produce temporary threshold shifts (TTS).

Tone-induced ERSPs

Fig. 2 shows the grand mean ERSP planes averaged across the 28 subjects for each of the 4 days. These have 3 main features:

(1) An augmentation in the 2–8 Hz range, peaking near 4 Hz soon after tone onset, followed by a longer-lasting diminution in the same frequency range.

(2) A ridge of 10–40 Hz augmented activity, with peak frequencies near 11 and 18 Hz, emerging during the latter portion of tone, and reaching a maximum shortly after tone offset.

(3) A suppression of EEG amplitude at the 39 Hz SSR driving frequency during tone presentation.

Note that the 11–18 Hz ridge of augmented activity appears to be smaller during free-field exposure (day 3) than on the other days. The actual difference between the ERSP planes for days 2 and 3 is shown in Fig. 3A.

To evaluate the consistency of ERSP differences across subjects, F statistics were computed separately for each time/frequency point in the ERSP planes by analysis of variance on ERSP data for each pair of days. To reduce the likelihood of false positive findings due to multiple comparisons, a minimum significance level of $P < 0.001$ was used. For the comparison between pre-exposure measures (day 2 versus day 1), there were no points of significant difference in the ERSP plane. By contrast, Fig. 3B plots a map of F statistics on ERSP amplitude differences between days 2 and 3. In this figure only significant F values are shown ($F (1, 27) > 13.5$, $P < 0.001$).

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[2] Amplitude/baseline amplitude normalization and mean log averaging provide one of several possible stochastic estimates of mean tendency in the distribution of response epochs. Median, arithmetic mean, and root mean square averaging might also be used to give somewhat different estimates. Averaging of event-related spectra can also be carried out without normalizing, producing an event-related spectrum transform. However, this can be dominated by noisy large-amplitude epochs in which the tendency for proportionate amplitude increase or decrease is obscured or absent.

Significant ERSP differences appear near 8 Hz, soon after tone onset, and near 11 and 18 Hz near tone offset. Note that although the F statistic map has sharply defined time-frequency features, the ERSP difference plane (Fig. 3A) shows that sizable differences in the grand means occur over a broad range of frequencies between 8 and 25 Hz, and that the most significant frequencies are at the two angular peak frequencies (near 11 and 18 Hz) visible in each of the 4 ERSP planes (Fig. 2).

Note also (Fig. 2D) that recovery in the ERSPs recorded during the day after exposure (day 4) appears to be incomplete. F statistic plots of ERSP differences between days 2 and 4 (not shown) imply continued reductions in spectral reactivity on the day after exposure at about 18 Hz near tone offset, and at about 8 Hz near tone onset. The 8 Hz difference does not occur at a peak of either of the ERSP planes.

Linear regression of ERSP amplitudes on time of testing revealed no significant trends over time at any point in the ERSP plane, either during the day of exposure (day 3) or on the day of recovery (day 4). Since time of exposure on day 3 (and time after exposure on day 4) varied across subjects from 3 to 11 h, this result suggests that the psychophysiological effects of exposure were not incremental over this time frame, and that the effects of exposure on the ERSP may have lasted longer than 11 h after the end of exposure.

Target-induced ERSPs

Fig. 4 shows grand mean ERSPs to detected targets. The structure of the target-induced ERSP planes is similar to that of the tone-induced ERSPs, although the target responses are larger (note the different scales used in Figs. 2 and 4). Both ERSP responses, to the 1 sec tones and the 25 msec tone pips, include two
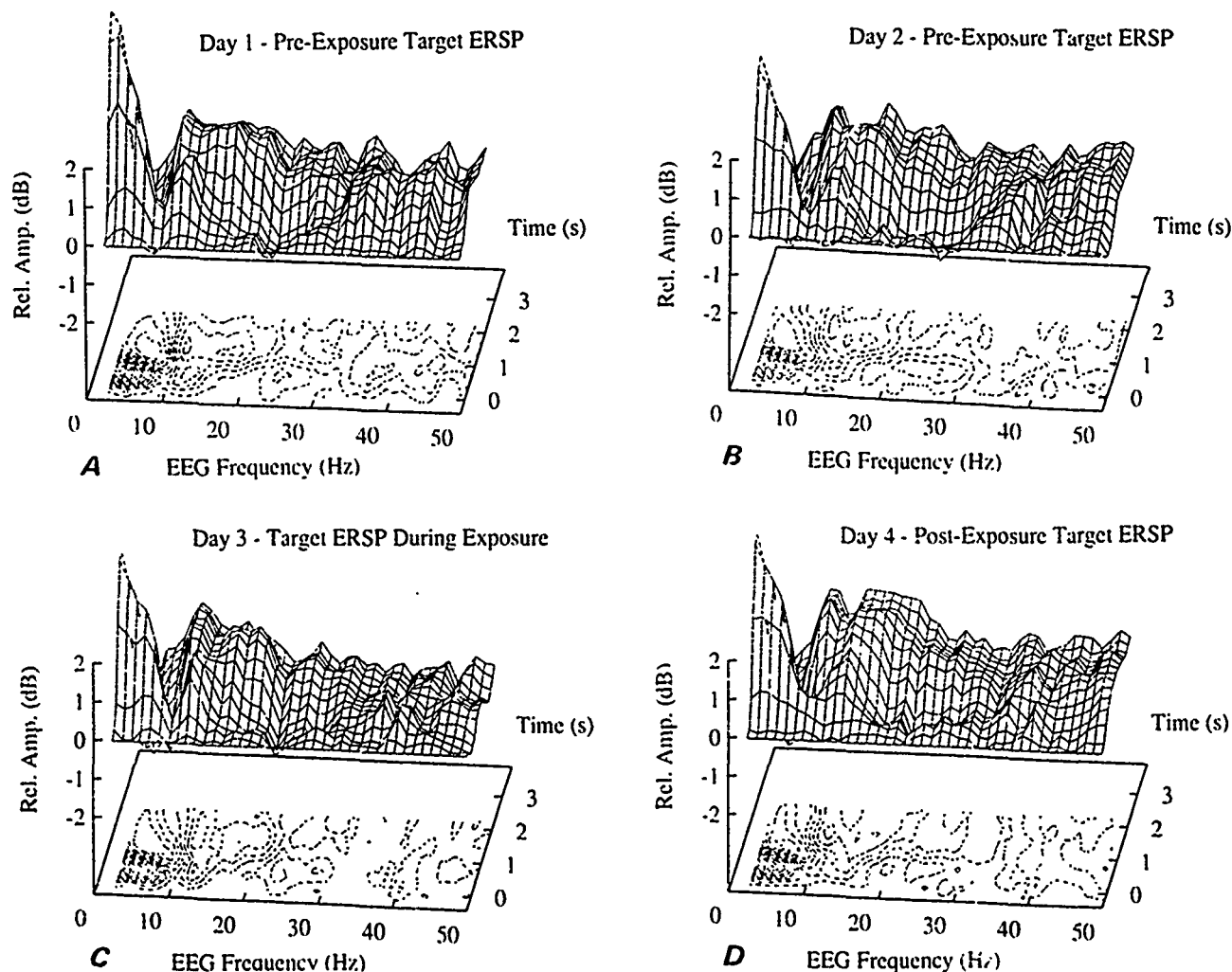

Fig. 4. ERSPs induced by occasional 25 msec target tone pips (at time 0) on days 1-4, grand means of responses from 28 subjects. Only detected targets not followed by any stimulus for 2.6 sec were averaged. Note the larger amplitude scale than in Fig. 2. No tone pip response differences were found between target ERSPs prior to and during exposure.

prominent regions of augmented activity, a peak enhancement at 2–8 Hz following stimulus onset, and a ridge of enhanced amplitude betwcen 10 and 25 Hz, peaking a second later. In all 4 responses, a band of diminished amplitudes between 20 and 30 Hz appears, peaking at about 500 msec, with apparent minima near 24 and 29 Hz. Target-induced ERSPs show little evidence of suppression of 39 Hz EEG following the target tone pips.

Note in Fig. 4 that, contrary to the tone-induced ERSP results, there is no apparent difference between the grand mean target-induced ERSPs on the day of exposure (day 3) and the other days. Statistical analyses confirmed that the (day 3 – day 2) difference was not significant ($P < 0.001$) at any point in the ERSP planes.

Discussion

Significance of the ERSP

Interest in event-related EEG dynamics other than those visible in the ERP has been heightened in recent years by increased appreciation of the extent of rhythmic activity induced in sensory cortices by stimulus events. These rhythms may be highly phase-coherent across selected spatially adjacent or non-adjacent cortical areas, but remain phase-incoherent across trials and thus may not appear in the averaged evoked response (Gray and Singer 1989; Llinás 1989; Murthy and Fetz 1992). Freeman and associates (Freeman 1975; Freeman and Skarda 1985), as well as others (Başar 1980; Grossberg 1980), have emphasized that in the process of perception, laminar brain structures, possibly including auditory cortex, may function as low-dimensional dynamic systems which match patterns of incoming stimulus-evoked activity against dynamically created and maintained impressional templates stored as a pattern of cell connectivities. In these networks, spatially coherent, narrow-band event-related oscillations may be a necessary concomitant of perception and object recognition, but still may not appear in time-domain average ERPs if their between-trials phase coherence is low.

The ERSP results obtained in this experiment indicate that the total brain electrophysiological response to auditory stimuli recorded from the human scalp is not fully revealed in the averaged ERP. In addition to the well-known time- and phase-locked auditory ERP components, auditory stimuli also produce time-locked but phase-incoherent changes in the EEG spectrum, which are revealed in the ERSP. In this experiment, wide-band auditory ERSPs induced by 1 sec tones and by 25 msec target tone pips shared several common features — numerically small (1–6 dB) but replicable enhancements of EEG amplitude time-locked to stimulus onset, particularly in the frequency ranges 2–8 and

10–40 Hz. In 2 frequency regions (8 and 11–18 Hz), tone-induced ERSP features were significantly depressed following sustained free-field sound exposure. Some further discussion of these results follows, organized by EEG band.

Delta and theta bands

ERSP effects below 2 Hz were not computed in these analyses, since accurate observation of low frequency ERSP activity requires using a longer transform. Near-DC ERSP activity, if demonstrated, might arise in part from variability in slow wave components of the ERP. Both the tone and target-induced ERSPs have a peak in the 2–8 Hz region near tone onset. This is the same time and frequency as the major peak in the spectrum of the auditory ERP (Makeig 1990; Pantev et al. 1991). Thus the early theta band ERSP peak must include the energy in the phase-locked ERP, possibly enhanced by contributions from trial-to-trial ERP variance. However, distinguishing between ERP variance and EEG amplitude modulation models requires an exact model of ERP variance, since otherwise either explanation (ERP variance or EEG modulation) can be used to fit any between-trial differences.

Alpha band

Although an alpha rhythm generated in the auditory cortex has recently been discovered, using biomagnetic sensors, and has been shown to be suppressed during mental tasks, this rhythm is not normally seen in electrical scalp recordings, since the standard electrode montage is not optimum for recording it (Kaufman et al. 1989). In this experiment, therefore, the lack of event-related EEG suppression near 10 Hz is not unexpected. Pfurtscheller and Klimesch (1991) also show an example of apparent augmentation at 10–12 Hz after offset of a visual stimulus, an effect they explain as relating to "disinhibition" in cortical fields. It is unclear whether the circa 11 Hz peak in the "ridge" feature of the ERSPs from these experiments represent enhancement of the subject's peak alpha frequency in the ongoing EEG, or whether it represents a separate, somewhat higher frequency phenomenon.

Beta band

Beta band EEG has long been assumed to represent engagement of cortical processing (Giannitrapani 1971), though specific models of the role and nature of this engagement have not been advanced. During exposure, suppression of the beta band augmentation was not uniform; the peak frequencies in the pre-exposure ERSPs (near 11 and 18 Hz) were also those most significantly reduced during exposure. Note that the prominent 11–18 Hz ridge of augmented activity occurring in these ERSP experiments was induced during performance of a simple task requiring subjects' atten-

tion to the stimuli. The ERSP enhancement was larger following targets than following tones, even though the tones had a longer duration and were perceived by subjects as being at least as intense as the tone pips. The 11–18 Hz augmentation might, therefore, be found to vary as a function of task and attention, an issue which will be tested in future experiments. If attention or other cognitive variables are shown to affect the beta band ERSP, this might suggest that human beta band activity has a function akin to that proposed for gamma band processes in animals (Gray et al. 1992). The 20–30 Hz basin of diminished amplitude in the target pip respon. es near 500 msec is in the range of the button press to these targets. This feature might therefore reveal movement-related suppression of coherent prestimulus beta band activity in some or all subjects (Bouyer et al. 1980).

Gamma band

In the human auditory cortex, the onset of tone or click stimuli generates a burst of 30–35 Hz of gamma band activity. The SSR represents primarily the overlapping sum of the phase-locked portion of these gamma band oscillations evoked in the auditory cortex (Makeig 1985, 1990; Mäkelä and Hari 1987; Pantev et al. 1991). The most prominent and consistent component in the tone-induced ERSPs (Fig. 2) is a suppression of EEG amplitude at the steady-state stimulus rate (39 Hz) during tone presentation. This no doubt represents the expected suppression of the steady-state respon. e (SSR) by the tone (Makeig and Galambos 1992a,b). Note, however, that the 39 Hz ERSP component did not differ between days 2 and 3 (Fig. 2B and C) and thus was not affected by the free-field exposure. This suggests that in these experiments, 3–11 h of free-field exposure did not alter the amplitude of gamma band evoked responses in the auditory cortex.

Gray and Singer (1989) have reported that a sizable subset of cells in the visual cortices of cats respond to moving visual stimuli with gamma band bursts of firing with a median frequency near 50 Hz. Murthy and Fetz (1992) have recently reported similar phase-coherent bursts near 30 Hz in the sensorimotor cortex of monkeys performing a difficult tactile task. These results parallel the phenomena long observed in the olfactory cortex, in which during inhalation of recognized odors, 40–90 Hz bursts appear (Freeman 1975). All these gamma band-induced rhythmicities have been theorized to provide a mechanism for rapid synchronization of brain activation of networks of cells responsive to different features of an object or event (so-called stimulus binding; Crick and Koch 1990). Freeman and Skarda (1985) and others (Bressler 1990) speculate that the occurrence of gamma band bursts is related to the meaning of a complex stimulus and relates specifically to the process of forming higher-order percepts.

In Fig. 4, it appears that the ridge of augmented 11–18 Hz activity near 1.5 sec is accompanied by a simultaneous, though smaller, increase in activity in the 20–40 Hz range and preceded by an amplitude trough peaking near 500 msec at 24 and 29 Hz, near the time of the button presses. Why do the target-induced ERSPs not reveal evidence of augmented higher frequency activity near stimulus onset (Makeig 1990)? It is likely that the time resolution of the spectral transform used was not sufficient to reveal the existence of bursts induced by target pips but with a much shorter duration than the 820 msec FFT windows used in the analysis. Future ERSP analysis might study this question using finer or variable time resolution methods (Przybyszewski 1991).

General discussion

The auditory ERSP

This study introduces a wide-band measure of event-related brain dynamics, the event-related spectral perturbation (ERSP), and demonstrates a new class of effects of exposure to sound on the brain. ERSPs induced by two very different auditory stimuli appear to have several time-frequency features in common, possibly reflecting the activity of common brain response systems. The similarities between the tone- and target-induced ERSPs in this experiment suggest that similar ERSP features may be induced by a wide variety of auditory stimuli. Note that the 1.0–1.5 sec latency of the beta band augmentation does not seem to depend on the duration of the stimulus and, therefore, in the tone-induced ERSPs it is not a response to tone offset. The early 2–8 Hz ERSP peak may represent, in part, spectral power in the ERP itself and, in part, trial-to-trial variability in the phase-locked ERP. The later beta band component is less likely to arise from response variability, since there are no corresponding peaks in the ERP spectrum in this time period. This component, therefore, may represent new or enhanced beta and gamma band oscillatory activity peaking a second or more after stimulus onset, i.e., later than the principal components of the ERP.

As the physiological origins and psychological concomitants of the ERSP components are not yet known, speculations about the meaning of differences in tone-induced ERSPs before, during, and after free-field exposure must be tentative. These differences cannot be due to habituation in the inner ear or peripheral auditory pathways, since these would most probably have been accompanied by changes in behavioral thresholds. Intensive audiometric testing performed during this experiment failed to reveal any effects of free-field exposure on auditory thresholds. This suggests that the exposure produced no lasting changes in

the peripheral auditory system, and no change in the information delivered to those central pathways serving signal detection.

Increasing time of exposure might be expected to produce increasing effects on auditory spectral reactivity. However, no significant linear trends in ERSPs were found for any day of testing. This suggests that 3 h of exposure (the minimum prior to testing for any subject) was sufficient to produce the ERSP changes observed on day 3. That some ERSP changes remained on day 4 (3–11 h after the end of exposure) also implies that reactive stress or arousal associated with the onset of exposure was not the cause of the changes observed in the tone-induced ERSPs on day 3, since stress might be expected to diminish on day 3, as subjects became habituated to their new environment, and to ebb rapidly on day 4 after exposure ended.

ERP / ERSP

The ERSP measures event-related perturbations in EEG amplitudes, but, as computed here, necessarily includes the spectral energy of the ERP itself [3]. To remove this confound, EEG epochs time-locked to the stimulus might first be averaged to form an ERP. This mean ERP could then be subtracted from the response epoch prior to analysis. However, removing the mean ERP from each epoch before spectral analysis would involve an implicit assumption that event-related brain dynamics can be modeled as the sum of a stable ERP and a reliable pattern of EEG amplitude modulation. Such a model would not take into account EEG phase-dependent interactions (Jansen and Brandt 1991), and further, the actual effect of subtracting the mean ERP from each epoch prior to the ERSP transform would be relatively small, particularly at frequencies above 10 Hz where auditory ERP amplitudes are 15 dB or more below mean EEG amplitudes.

While the ERSP reveals new and potentially important information about event-related brain dynamics, it cannot reveal interactions between the ERP amplitude, latency variability, and EEG spectral modulation [4]. Removing a mean ERP from each ERSP epoch prior to analysis would not eliminate the effects on the ERSP of trial-to-trial variability in the ERP itself. For example, a skewed distribution of between-trial ERP

variabilities could also create the appearance of augmented EEG amplitude in an ERP-corrected ERSP. These complexities cannot be resolved by ERSP analysis alone without detailed models for ERP variance and EEG modulation. Mixed models in which the phase coherence of evoked response components is neither one (phase locked ERP) nor zero (independent EEG) are also possible. In such models the relationship between pre- and post-event EEG phase could also be perturbed by an experimental event (Jansen and Brandt 1991; Achimowicz 1992), and this could produce additional ERSP effects.

Further questions

The sensitivity of the tone-induced ERSP to prior stimulation, in the absence of peripheral effects, suggests that ERSP measures might prove sensitive to the effects of previous experience on brain dynamics. A number of further questions are raised by the ERSP results of this study:

(1) Prior to this experiment, data from a group of 10 subjects were collected using an exposure and intensity of 83 dB (A-weighting). The ERSPs for this group were generally similar to those reported here, as were the differences between the ERSPs before and during exposure. However, for the 83 dB group these changes were smaller and less significant, possibly because of the smaller number of subjects. Little else is known about the effects of stimulus intensity on the ERSP.

(2) The tone frequencies used for evoked response testing were the same as those used in free-field exposure. It is not known whether ERSPs to tones of different frequencies would also be suppressed during exposure.

(3) Throughout these experiments subjects were attending the tones in anticipation of responding to the occasional targets. The ERSPs induced by target pips were larger than those induced by tones, possibly on account of their comparative probability or task relevance. Effects of alertness, task relevance, and attention on ERSP form and amplitude remain to be explored (Makeig and Inlow 1993).

(4) Averaging over all subjects and trials yields information about only the most global event-related response patterns — between-subject and between-trials differences are ignored. Gray et al. (1992) have recently shown that spatial coherences among event-related gamma band oscillations in the visual cortex of cats are complex transitory dynamic phenomena. It is probable that single-trial ERSPs reported here also represent transient and somewhat variable dynamic events. Cluster analysis of subjects and trials, followed by discrete or moving subaverages, might prove useful for better understanding the brain processes measured by ERSPs.

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[3] This confound between EEG and ERP spectral characteristics is also present in narrow-band ERDs.

[4] In general, vestiges of phenomena revealed by ERSP analysis can remain in ERPs as time-dependent changes in the spectral content of the background EEG noise remaining in the ERP average. However, as ERP analysis is designed to remove phase-incoherent activity occurring in the response period, the vestiges of the ERSP remaining in the ERP are normally small, and in general ERSP features cannot be expected to be recaptured efficiently from the ERP by post hoc spectral analysis.

Conclusions

(1) A wide-band, normalized measure of event-related spectral reactivity, the event-related spectral perturbation or ERSP, can be used to measure aspects of event-related brain dynamics not found in the ERP.

(2) Auditory ERSPs had similar forms and latencies for two very different inducing stimuli — 1 sec, low-frequency tones, and 25 msec, 3 kHz tone pips. A prominent feature of both ERSPs was a prominent, relatively late (1.0–1.5 sec) ridge of augmented 11–18 Hz EEG amplitudes which was larger in response to the target tone pips.

(3) Some features of the auditory ERSP appear to be sensitive to previous sound exposure. In this experiment, some features of the tone-induced ERSPs were significantly suppressed following prolonged exposure to moderately loud pure tones and appeared to not recover fully on the day following exposure.

The physiological significance of the ERSP features revealed in these experiments is not yet known, and many experiments may be required to determine their properties. Auditory, visual and somatosensory stimulus-induced ERSPs may be expected to differ in form, time course, and scalp distribution. Analysis of single response epochs, multi-channel source localization studies, and multi-modality experiments would also be of interest. Results of cognitive experiments, including experiments involving omitted stimuli, could add to our understanding of the relation of EEG activity to cognition. It is likely that further research using wide-band ERSP measures can yield new information· on brain dynamics of perception and cognition.

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A new measure of event-related brain dynamics, the event-related spectral perturbation (ERSP), is introduced to study event-related dynamics of the EEG spectrum induced by, but not phase-locked to, the onset of the auditory stimuli. The ERSP reveals aspects of event-related brain dynamics not contained in the ERP average of the same response epochs. Twenty-eight subjects participated in daily auditory evoked response experiments during a 4 day study of the effects of 24 h free-field exposure to intermittent trains of 89 dB low frequency tones. During evoked response testing, the same tones were presented through headphones in random order at 5 sec intervals. No significant changes in behavioral thresholds occurred during or after free-field exposure. ERSPs induced by target pips presented in some inter-tone intervals were larger than, but shared common features with, ERSPs induced by the tones, most prominently a ridge of augmented EEG amplitude from 11 to 18 Hz, peaking 1-1.5 sec after stimulus onset. Following 3-11 h of free-field exposure, this feature was significantly smaller in tone-induced ERSPs; target-induced ERSPs were not similarly affected. These results, therefore, document systematic effects of exposure to intermittent tones on EEG brain dynamics even in the absence of changes in auditory thresholds.

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